DETAILED ACTION

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 33, 34 and 44-47 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-3 of U.S. Patent No. 7,264,942 in view of Borasio et al. (1998; cited in the IDS filed 6/14/05). The instant claims are drawn to a method for assessing a compound's ability to specifically inhibit JNK wherein the method requires the assessment of compounds to inhibit JNK in three types of experiments: in vitro, measuring apoptosis in cultured neuronal cells and in a mammal. The data from the steps are correlated to assess the ability of the compound to specifically inhibit JNK (page 1439, last paragraph).

The claims of '942 are also drawn to a method for assessing the ability of a compound to specifically inhibit JNK by carrying out the first two steps of the instantly claimed invention. Thus, the claims of '942 lack the third step (e.g., mammalian testing and correlation of data from testing in vitro and with cultured neuronal cells).

Borasio discloses that it is desirable to identify compounds which can decrease the activity of JNK in neurons for the treatment of neurodegenerative disorders (abstract). Borasio discloses the effect of test compounds on JNK/apoptosis in cultured neuronal cells. Borasio asserts that the development of small trophic molecules that can cross the blood-brain barrier might represent an alternative therapeutic approach, compared to the use of problematic polypeptide trophic factors, for the treatment of neurodegenerative diseases. Therefore, Borasio clearly establishes the desirability of correlating results of test compounds on JNK activity of neurons in cultures to the treatment of mammals having neurodegenerative diseases.

It would have been obvious to one of ordinary skill in the art at the time the invention was made to add a mammalian testing step of compounds identified by the in vitro and cultured neuronal cells steps of the claims of '942 and to correlate the data from all testing methods. The ordinary artisan would have been motivated to do so because Borasio establishes the desirability testing potential JNK-inhibiting compounds in neural cells to identify compounds to treat neurodegenerative diseases in humans. The ordinary artisan would have been motivated to use an animal model to assess the in vivo effect because it is standard practice in the pharmaceutical industry to initially test compounds with animal an model prior to human testing trials. The ordinary artisan

would have had a reasonable expectation that one could successfully add in vivo testing to the method of '942 because it is well known to the ordinary artisan to correlate multiple levels of testing of potential pharmacologically active compounds (e.g., in vitro, neuronal cell culture, in vivo) to determine new disease treatment.

Terminal Disclaimer

The terminal disclaimer filed on 7/7/08 disclaiming the terminal portion of any patent granted on this application which would extend beyond the expiration date of U.S. Patent No. 7,264,942 has been reviewed and is accepted. The terminal disclaimer has been recorded.

EXAMINER'S AMENDMENT

An extension of time under 37 CFR 1.136(a) is required in order to make an examiner's amendment which places this application in condition for allowance. During a telephone conversation conducted on July 3, 2008, Basil Krikelis requested an extension of time for THREE MONTH(S) and authorized the Director to charge Deposit Account No. 50-3570 the required fee of $1,050.00 for this extension and authorized the following examiner's amendment. Should the changes and/or additions be unacceptable to applicant, an amendment may be filed as provided by 37 CFR 1.312. To ensure consideration of such an amendment, it MUST be submitted no later than the payment of the issue fee.

The application has been amended as follows:

IN THE CLAIMS:

Claim 33 was replaced by the following:

-- 33. A method for assessing a compound's ability to specifically inhibit c-Jun kinase (JNK) activity in a mammal susceptible to or having a neurological condition, comprising:

(a) incubating in vitro said compound in the presence of a JNK substrate and an isolated JNK, under conditions sufficient for kinase activity;

(b) determining the amount of the JNK substrate that has been phosphorylated by JNK, wherein a decrease in the amount of the phosphorylated JNK substrate, when compared to incubating the isolated JNK with the JNK substrate in the absence the compound, is indicative of the compound's ability to inhibit the JNK activity;

(c) contacting the compound having an ability to inhibit JNK activity from step (b) with neuronal cells having JNK activity that have been transfected with a mutated protein or treated with a neurotoxin to induce apoptosis, wherein the mutated protein comprises polyglutamine stretch-expanded huntingin or C-terminal 100 amino acids of amyloid precursor protein;

(d) comparing the occurrence of apoptosis in the neuronal cells in the presence and the absence of the compound from step (b) wherein a decrease in the occurrence of apoptosis in the neuronal cells in the presence of the compound when compared to the occurrence of apoptosis in the neuronal cells in the absence of the compound is indicative of the ability of the compound to prevent neuronal cell death;

(e) administering to a mammal susceptible to or having a neurological condition the compound from step d), having the ability to inhibit JNK in vitro in step (b) and to prevent neuronal cell death in step (c), under conditions sufficient to allow for proper pharmacodynamic absorption and distribution thereof in the animal;

(f) harvesting a neuronal tissue sample from the mammal; and

(g) determining apoptosis in the tissue sample;

(h) correlating the results of steps (d) and (g), wherein a decrease in apoptosis in the neuronal tissue sample, when compared to apoptosis in a neuronal tissue sample from the mammal not administered the compound, as determined in step (g), and wherein the occurrence of apoptosis in the neuronal cells in the presence of the compound is less than the occurrence of apoptosis in the neuronal cells in the absence of the compound, as determined in step (d), taken together, correlate with the compound's ability to specifically inhibit JNK kinase activity in a mammal susceptible to or having a neurological condition. --

Any inquiry concerning this communication or earlier communications from the examiner should be directed to SUSAN HANLEY whose telephone number is (571)272-2508. The examiner can normally be reached on M-F 9:00-5:30.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Michael Wityshyn can be reached on 571-272-0926. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Sandra Saucier/
Primary Examiner, Art Unit 1651

/Susan Hanley/
Examiner, Art Unit 1651

Search Notes (continued)

Application/Control No.	Applicant(s)/Patent under Reexamination	
10/042,614	LIU, YA FANG	
Examiner	**Art Unit**	
SUSAN HANLEY	1651	

SEARCHED			
Class	Subclass	Date	Examiner

INTERFERENCE SEARCHED			
Class	Subclass	Date	Examiner

SEARCH NOTES (INCLUDING SEARCH STRATEGY)		
	DATE	EXMR
WEST: DWPI, USPAT, EPAB, JPAB, PGPUBS, USCOR, see attached	7/2/2008	SMH